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L5: Entry 1 of 1 File: USPT May 2, 2000

DOCUMENT-IDENTIFIER: US 6056973 A
TITLE: Therapeutic liposome composition and method of preparation

BSPR:
Liposomes, spherical, self-enclosed vesicles composed of amphipathic lipids, have been widely studied and are employed as vehicles for in vivo administration of therapeutic agents. In particular, the so-called long circulating liposomes formulations which avoid uptake by the organs of the mononuclear phagocyte system, primarily the liver and spleen, have found commercial applicability. Such long-circulating liposomes include a surface coat of flexible water soluble polymer chains, which act to prevent interaction between the liposome and the plasma components which play a role in liposome uptake.

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L3: Entry 1 of 9 File: USPT Dec 7, 1999

DOCUMENT-IDENTIFIER: US 5999678 A
TITLE: Laser delivery means adapted for drug delivery

DEPR:
Cancer therapy can proceed along several different lines, all of which seek to kill or limit the growth of cancer cells while doing minimal damage to the host. Thus, any difference in cancer cell properties (e.g. metabolism, cell-surface antigen presentation) from healthy host cells is a target for exploitation. With the local administration of therapeutics, these differentiating factors may be created and/or exploited. For example, the local administration of cytotoxins or growth inhibitors may allow higher local concentrations of the compounds than would be achievable by systemic administration. Differences in cell-surface recognition molecules may be a site for antibody therapy. Differences in tumor morphology are also potential sites of intervention: for example, anti-VEGF may be useful in retarding the vascularization of the interior of a solid tumor, thereby slowing its growth rate. Some examples of agents in the category include:

DETL:

Agent Category Name	Manufacturer	Indication	Form
Adjuncts	Kytril Smith-	prevention of nausea and IV (grani-	Kline
vomiting associated with setron	Beecham	emetogenic cancer therapy,	HCI)
including high-dose cisplatin	Androgen	Lupron	TAP
palliative treatment of IM	Inhibitors	(leupro-	Pharma-
prostatic cancer	lide	ceuticals acte-	tate)
Antibiotic	Doxo-	Astra USA	produces
regression in IV Deriva-	rubicin	disseminated	neoplastic
tives Hydro-	conditions and possibly	chlor-	some
solid tumors	ide	Anti-	Nolva-
Zeneca treatment of metastatic oral	estrogen	dex	Pharma-
breast cancer (ta-	ceuticals	moxi-	fen ci-
trate)	Anti-	Rofer-	Roche
treatment of hairy cell IM/	metabo-	on-A	leukemia
and AIDS-	SC	lites (inter-	related
Kaposi's	feron	sarcoma	alfa- 2a)
Cytotoxic	Taxol	Bristol-	treatment
of metastatic IV	Agents	Myers	carcinoma
of the ovary and	Squibb	treatment of	breast
cancer	Enzyme	Ras	Genentech
treatment of pancreatic	pre-	Inhibitors	far-
and colon cancers	clini-	nesyl-	cal
trans-	fer-	ase	inhibi-
tor (pre-	clini-	cal)	Hormones
Depo-	Upjohn	adjunctive	therapy
and IV	Provera	palliative	treatment
of (med-	inoperable,	recurrent,	roxy-
and metastatic	endo-	proges-	metrial
or renal	carcinoma	terone acetate)	Immuno-

myeloma oral Deriva- pha- tives lan HCI)

URNM:
Isner et al.

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L4: Entry 1 of 31 File: USPT Jun 6, 2000

DOCUMENT-IDENTIFIER: US 6071493 A
TITLE: Method of screening for an agent that inhibits mononuclear phagocyte-plaque component complex formation

DEPR:
Agents detected by a process set forth herein for the detection of inhibitor of a mononuclear phagocyte-plaque component complex formation were in fact found to be inhibitory to complex formation. One plaque suppressor agent is identified herein as SEQ ID NO: 1, HHQK. Agents comprising HHQK, HHQK-like agents having HHQK activity such that they are inhibitory to complex formation include agents having a secondary or tertiary structure substantially similar to HHQK. Alternatively, HHQK-like agents or activity may be measured by similar hydrophobicity, hydrophilicity, acidity, and basicicity of the agent or side chains thereof. In addition, minor variations in the measurements identified herein are considered to be substantially similar to HHQK. Another plaque suppressor is heparan sulfate and heparan sulfate-like agents having heparan sulfate activity such that they are inhibitory to complex formation include agents having a secondary or tertiary structure substantially similar to heparan sulfate. Alternatively, heparan sulfate-like agents or activity may be measured by similar hydrophobicity, hydrophilicity, acidity, and basicicity of the agent or side chains thereof. In addition, minor variations in the measurements identified herein are considered to be substantially similar to heparan sulfate. A composition comprising heparan sulfate or a heparan sulfate-like agent is provided herein, such that the composition is provided in a pharmaceutically acceptable carrier in a therapeutically effective amount. Suitable pharmaceutical carriers are well known in the art and described, for example, in Gennaro, Alfonso, ed., Remington's Pharmaceutical Sciences, 18th Edition, 1990, Mack Publishing Co., Easton Pa., a standard reference text in this field. The particular amount of the compositions of the invention that will be administered to the mammal for any particular condition will depend on the type of illness, and other factors such as the weight and age of the patient and route of delivery.

DEPR:
Agents which inhibit plaque component induced neurotoxicity of a mononuclear phagocyte are referred to as inactivators of neurotoxic mononuclear phagocytes. One inactivator agent is identified herein as chloroquine. Agents comprising chloroquine and chloroquine-like agents

similar to chloroquine. Alternatively, chloroquine-like agents or activity may be measured by similar hydrophobicity, hydrophilicity, acidity, and basicicity of the agent or side chains thereof. In addition, minor variations in the measurements identified herein are considered to be substantially similar to chloroquine. A composition comprising chloroquine or a chloroquine-like agent may be used in a similar therapeutic and pharmaceutic manner as set forth above for the plaque suppressor agents.

DEPR:
Agents which inhibit plaque component induced neurotoxicity of a mononuclear phagocyte are referred to as a neurotoxic blocker. One blocker agent is identified herein as tyramine. Agents comprising tyramine and tyramine-like agents having tyramine activity such that they are inhibitory to neurotoxin effects on neurons, include agents having a secondary or tertiary structure substantially similar to tyramine. Alternatively, tyramine-like agents or activity may be measured by similar hydrophobicity, hydrophilicity, acidity, and basicicity of the agent or side chains thereof. In addition, minor variations in the measurements identified herein are considered to be substantially similar to tyramine. A composition comprising tyramine or a tyramine-like agent may be used in a similar therapeutic and pharmaceutic manner as set forth above for the plaque suppressor agents.

DEPR:
The present invention offers strategies for intervention in the pathology resulting from neurotoxic microglia in AD including (1) suppression of signaling steps as neuritic/core plaques turn quiescent microglia into reactive ones, (2) inhibition of microglial synthesis and secretion of neurotoxins, and (3) the blockade of neurotoxin attack upon neurons. In pursult of the first of these strategies, specific domains of A.beta. responsible for the various steps in the A.beta. induced cascade of cellular response leading to neurotoxic microglia may be manipulated. Since the cell attachment domain in the N-terminal portion of A.beta. is not itself toxic, induction of neurotoxic microglia by competition with small AD peptides may be blocked. Indeed, while anti-inflammatory drugs have been recommended as beneficial for AD (Breitner et al., 1990; McGeer et al., 1990; Schnabel, 1993; Eikelenboom et al., 1994; Lucca et al., 1994), the recommended drugs could not be properly assayed until the invention of the screening methods provided herein. In addition, microglial suppressants such as chloroquine are also indicated as a likely candidate (Giulian et al., 1989; Giulian and Robertson, 1990), since commonly used immuno-suppressants (including glucocorticoids) do not reduce neurotoxic activities of brain mononuclear phagocytes (Giulian, 1992). Finally, the neurotoxin secreted by plaque-activated microglia can be blocked by antagonists of the NMDA receptor. NMDA receptor antagonists useful for stroke, trauma, and epilepsy might now be screened for AD and may ultimately offer benefit to the AD patient. Inhibiting the A.beta. activation of microglia offers a number of therapeutic